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In Our Opinion

NUTRITIONAL FACTORS RELATING TO GINGIVAL AND PERIODONTAL HEALTH AND DISEASE.

Initial Statement

HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

PERIODONTOCLASIA is due to inflammation at the base of the gingival sulcus which involves the connective tissue of the cemental gingiva. Capillaries are plugged by the inflammatory reaction thus leading to partial stasis, deficient oxidation, and the accumulation of toxic and acid end products. The effect of these chemical changes is swelling, final dissolution of the principal fibers of the cemental gingiva with proliferation of epithelium down along the cementum to take its place. Splitting of the loose epithelium or actual penetrating ulcers at the base of the gingival sulcus create periodontal pockets. The cause of these changes is bacterial accumulations next to and under the marginal gingiva combined with insufficient function in the marginal gingiva to maintain its tone, its blood supply, and its resistance. The condition is greatly aggravated by traumatic pressures on the pericementum that injure that tissue and alter the blood supply through it. Essentially, gingivitis and periodontoclasia are local diseases related to bacterial accumulations, local function, and local blood flow.

Systemic conditions modify this process in many ways some of which are known and many as yet unknown. Every periodontist has seen cases in which there was every local reason for gingivitis and periodontoclasia to be present yet the gingival tissues were healthy. Also, he has seen cases in which the most exacting local treatment and home care brought health only very slowly or incompletely.

The chemistry of resistance to infection is by no means well developed. Many factors undoubtedly play a part in it such as the inherited chemical constitution, antibodies, oxidation reduction enzyme system, permeability of capillaries, quality of squamous cell epithelium, and others. These are influenced by the presence or deficiency of vitamins, minerals, or endocrine hormones, the presence of toxins and poisons in the blood, excess sugar, various blood diseases, neurovascular disturbances, lack of local tissue function, and probably many other abnormal systemic conditions. Fatigue, mental stress, and other unhygienic conditions effect resistance to infection through chemical mechanisms, the nature of which is not completely understood.

The dietary factors about which we have some knowledge can be fairly briefly summarized.

There are three known systemic conditions which alter the periodontal tissues so as to lead to loosening of the teeth and bone loss prior to gingival inflammation. These are scurvy, osteitis fibrosa cystica, and a highly alkaline diet. Vitamin C deficiency leads to failure in formation and maintenance of intercellular connective tissue substance. Capillaries become fragile, connective tissue fibers loosen, and bone resorbs easily under stress. Boyle (*J. A. D. A.* 25:1436. 1938) states that, "Vitamin C deficiency is the only nutritional deficiency which in our experience produces the characteristic features of the systemic type of

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pyorrhea in experimental animals." By systemic type he means the diffuse alveolar atrophy as described by Gottlieb in which there are deep bone losses prior to bacterial invasion and pocket formation, and in which teeth shift from stresses that normally would not cause shifting.

Frank scurvy in present day Americans is quite uncommon and when it appears, the signs are subepithelial hemorrhages, old rose color of gingiva, hypertrophy of interdental papillae, and gingival bleeding before signs of pericemental and alveolar breakdown appear. The cases which might be diagnosed as diffuse alveolar atrophy so far recorded have not proved to be scorbutic, and demonstrably scorbutic cases in our experience have not shown diffuse alveolar atrophy as an early symptom.

Osteitis fibrosa cystica is due to overactivity of the parathyroid gland which upsets the balance of ionizable and combined calcium and phosphate with loss of bone. Cystic cavities in bone develop which may involve teeth and cause their loosening from lack of alveolar support.

Jones and Simonton (*J.A.D.A.* 15:881, 1928) gave sodium carbonate to dogs with resulting losses of calcium and phosphate from the body. This caused demineralization of bone including the alveolar bone. They regard an alkaline ash diet as the cause of diffuse alveolar atrophy. McCollum *et al* (*Newer Knowledge of Nutrition,* 5th edition, 1939) says that a basic ash or acid ash diet due to food alone is not significant in calcium and phosphate metabolism. There is no reason to expect alveolar bone to be more sensitive than other bone in any general demineralization disease. In the absence of local irritants, it is usually more resistant than the long bones. General demineralization has never been demonstrated in the cases diagnosed as diffuse alveolar atrophy. The case for alkaline ash diet acting through bone metabolism to cause periodontoclasia needs much more proof than there is at present.

There are probably other diseases which have a similar effect on periodontal connective tissue and bone, but these are at present unknown and almost certainly are not directly due to nutritional faults. Diabetes has some deleterious effect on blood capillaries, but most of its ill effects on the periodontium are by lowering resistance to staphylococcus infection. Leukemia and purpura hemorrhagica cause gingival bleeding and hypertrophy, but do not appear to alter the underlying periodontal structures. Magnesium deficiency, in its later manifestations prior to death in experimental animals, causes a great hyperplasia of the connective tissue of the gingiva so as practically to bury the molar teeth. Dilantin sodium medication often causes hypertrophic gingivitis without relation to accumulations on the teeth. Pregnancy gingivitis is due to hormonal disbalance acting on the gingival epithelium. None of these diseases can account for periodontal deterioration prior to bacterial invasion of the gingiva.

Colyer (*Variations and Diseases of the Teeth of Animals,* London, 1936) has made an extensive study of periodontoclasia, on the teeth of animals, wild and domestic, free and captive. He finds that it is very common in many animals and a frequent cause of premature death. In animals in the free state, it is due to foreign bodies which become impacted under the gingiva and start the train of infection. In animals fed by man, the disease is due to pappy food which fails to cleanse the teeth and massage the gingiva and leaves a residue on the tooth surfaces to cause gingivitis. He says he has never seen a case in which the condition could be accounted for by underlying tissue disease changes prior to the surface infection or injury.

This brings us to the nutritional causes of excessive bacterial growth on the teeth and lack of resistance to their presence.

1. Overeating, just prior to a period of rest, tends to globulin overflow in the saliva leading to accumulations on the teeth which act as a medium for calculus formation and certainly increase bacterial activity (G. V. Black, *Special Dental Pathology,* 1915, pp. 73-98). Some patients have this salivary protein deposit who do not overeat. The cause for this is unknown. Prinz and Greenbaum (*Diseases of the Mouth and Their Treatment,* 2nd edition, 1938, p. 472) say, "We have observed individuals develop a heavily furred tongue while on an 'alkaline' (milk and vegetable) diet and have it disappear on an 'acid' (meat) diet." This is probably related to the salivary soft deposits on the teeth.

2. Vitamin A deficiency acts primarily on epithelial tissue. Susceptibility to infections in the mouth results from changes in epithelium. Mellanby (*Diet and Teeth,* Part

2A, 1930) has described gingival lesions which led to periodontoclasia in dogs deficient in vitamin A. King *(British D. J.* 68:349, 1940) has produced similar lesions in rats. Boyle *(loc. cit.)* on the other hand has not seen this effect on his A deficient animals. It would seem that such an effect is rather a late stage in extreme A avitaminosis. Certainly from clinical observation, vitamin A deficiency can account for but few cases of low gingival resistance.

3. Thiamin affects tissue respiration and while it does not appear to affect the resistance to infection mechanisms in normal gingival tissue, a deficiency may adversely influence the tissue metabolism in congested gingival tissue.

4. Nicotinic acid (niacin) avitaminosis is believed to be the cause of pellagra in which Vincent's-like necrotic ulcerations of the gingiva and mucosa are common. King *(Lancet,* 2:32, 1940) has treated a considerable series of Vincent's infection cases with nicotinic acid without local treatment and obtained prompt and complete recovery. Several dentists in the United States whose work is so far unpublished have obtained similar results. Animal experiments with niacin avitaminosis in monkeys and dogs, almost regularly produce Vincent's gingivitis and sometimes noma. Vincent's infection has been produced in animals on diets deficient in vitamin A, C, or D and supposedly protected in other respects, but mostly it is associated with niacin deficiency. It would appear that Vincent's infection is definitely a symptom of nutritional deficiency and that when present, the diet should be regulated and at least temporarily fortified with vitamin administration. All authorities on the B group of vitamins stress the inter-relationships of vitamins. Vitamin deficiencies are seldom single. A deficiency of one enhances the ill effects of a deficiency of another. There is clinical evidence that periods of mental stress, and other unhygienic conditions like confinement or overcrowding, increase the tendency to Vincent's infection. Such conditions possibly increase the wastage and therefore the need for some of the vitamins.

5. The case for vitamin D is by no means clear. It regulates the proportions of calcium and phosphate in the blood and thereby the absorption and elimination of these nutrients. In periods of great calcium phosphate need, as in growing children and pregnant and nursing mothers, vitamin D administration is a necessity unless both calcium and phosphate are abundant and well proportioned in the diet.

Its lack leads to rickets in young children and osteomalacia in adults. Phosphate is evidently often deficient in the nutrition as shown by the prevalence of rickets, osteomalacia, milk fever, and other phosphate deficiency diseases prior to the general use of cod liver oil. Unless milk products and eggs are present the diet may well be deficient in phosphate for the phosphate contained in cereal grains is not well utilized.

The animal organism commonly gets vitamin D from skin absorption or by ingestion of sterols, irradiated by sunlight, which are secreted by the skin and cover hair and feathers. Washing with soap and water removes these secreted sterols. Also, irradiation by sunlight is slight for many people in many regions especially in the winter season. Yet bone disorders attributable to this cause, excepting in young children and pregnant or nursing mothers, seem to be relatively uncommon. Boyle *(loc. cit.)* did not find D avitaminosis to be associated with alveolar bone disturbance in his animals. The relation of D avitaminosis to bacterial resistance in general, and especially in the gingiva, has not been satisfactorily demonstrated. Clinically there appears to be no direct correlation.

6. Mild C avitaminosis as a cause of lowered resistance of the gingiva has considerable confirmation. Hanke *(Diet and Dental Health,* 1933) was able to overcome gingivitis in a large group of orphanage children without change in their mouth care, simply by adding relatively enormous amounts of citrus fruit juice to the diet over a period of a year. When these children had the orange and lemon juice reduced to three ounces daily, the gingivitis recurred in many of them. That C avitaminosis is associated directly with gingivitis has been confirmed in much more complete tests conducted on school children in New York City under the auspices of the Milbank Memorial Fund *(The Changing Front of Health,* 1940, pp. 49-50). There is considerable evidence that vitamin C is related to the immunological mechanism (McCollum *et. al., loc. cit.)* and resistance to infection.

According to the Food and Nutrition Board of the National Research Council, the dietary requirements for a moderately active man of 154 lbs. and a woman of 123 lbs.

are respectively:

	CALORIES	PROTEIN	CALCIUM	IRON	VIT. A	THIAMIN	RIBO-FLAVIN	NIACIN	VIT. C	VIT. D
		gm.	*gm.*	*mg.*	*IU*	*mg.*	*mg.*	*mg.*	*mg.*	*IU*
Man	3000	70	0.8	12	5000	1.8	2.7	18	75	400
Woman	2500	60	0.8	12	5000	1.5	2.2	15	70	400

(*Handbook of Nutrition* Pub. by Am. Med. Ass'n 1943)

It requires rather skillful designing of the menu to procure a diet which is optimum in all respects. Besides the amounts in the original foods, losses must be considered from storage, preparation, cooking, and individual failures in digestion, absorption or metabolism. Surveys of school children in poorer district schools and from private schools in New York City (Milbank Fund *loc. cit.*) show that a large proportion of children are deficient in one or more of each of these nutrients, many in the low income group not getting even a fourth of the optimal amount of some.

The excessive consumption of sugar and retention in the mouth of candy such as lifesavers act not only to increase caries of the teeth, but also to increase the bacterial growth which often causes gingivitis. Besides excess sugar acts systemically by requiring extra amounts of phosphorus and thiamin for its metabolism (Everett, *Medical Biochemistry*, 1942, p. 599), while it decreases the amount of protective factors actually ingested by decreasing the appetite for good food.

In reviewing this subject, it is apparent that in humans as in animals, the chief dietary relation to gingivitis and periodontoclasia lies in the lack of self-cleansing effects of food. Accumulations of food or salivary deposits from some nutritional conditions lead to gingivitis and from that to periodontoclasia. Resistance is lowered to the Vincent's organisms apparently by subclinical niacin deficiency. Besides niacin, mild vitamin deficiencies in A, possibly D, and certainly C show evidence of influence in lowering resistance to bacterial accumulations on the teeth. The excessive use of candy and sweets aggravate accumulations of bacteria on the teeth and deficiencies of vitamins, minerals, and proteins. As regards primary underlying connective tissue changes, severe scurvy, alkaline medication, and hyperparathyroidism are known at present. Others remain to be discovered but will probably be found to be hormonal or toxic conditions and not nutritional excesses or deficiencies.

Discussion

HERMANN BECKS, M.D., D.D.S.,
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The Initial Statement by Dr. Leonard probably represents best the general level of knowledge in the field of nutrition of our general practitioners and paradentic specialists inasmuch as it contains very little factual information of experimental or clinical evidence which has appeared from time to time in the international literature during the last 10 years. I may even go further and express my keen disappointment about the first paragraph of this Initial Statement which leaves the impression that periodontoclasia is solely due to inflammation of the subepithelial connective tissue, while the paradental bone structure, as one of the most important tissues of the tooth supporting apparatus and which responds very readily to nutritional and metabolic disorders, is not even mentioned. I beg to disagree profoundly with this concept of periodontoclasia (if this should be the same pathologic condition which we term "paradentosis") because from a clinical, as well as experimental, point of view there should be no doubt any more in the mind of any biologically thinking dentist or paradental specialist that many constitutional factors, systemic diseases of various types, metabolic disorders, vitamin and mineral deficiencies, etc. can produce pathologic changes of varying degrees in paradental structures which are not all of an inflammatory type. I am sure that I will find myself in contrast to a large number of practicing members of the American Academy of Periodontology by stating that in our experience the systemic etiologic factors surpass by far in frequency those of local origin. Only very few systematic

checks have been made on systemic relationships; however, I would venture that in the neighborhood of 75 % or more, systemic factors are responsible for the development of paradentosis. Is it not common practice to recall patients with paradental diseases every six months? Do we not admit by this practice that we have not removed the etiological factor, and that we *expect* the condition to reoccur during the next few months? How many cases are permanently cured or arrested by *one* subgingival curettage or *one* electrocoagulation, or *one* gingivectomy? How many periodontists think in terms of assessing the *etiological background* of each individual case? And how many practitioners take into account nutrition as a possible causative factor?

The clinical experience of temporary favorable response to local treatment is by no means an indication that the etiology was of a local nature. With the newer knowledge of the oral aspects of certain blood dyscrasias (leukemias, agranulocytosis, etc.) and developments in vitamin B research with special reference to niacin deficiencies, our greatly exaggerated concept of the local role of so-called Vincent's microörganisms in the ulcerative type of gingivitis or stomatitis has been greatly shaken, and in many instances has the failure to recognize the systemic relationship to ulcerative lesions in the mouth led consequently to deep pocket formation and loss of teeth.

For these reasons I cannot agree with the concluding sentence of the first paragraph: "Essentially gingivitis and periodontoclasia are local diseases related to bacterial accumulations, local function, and local blood supply." This statement implies that the oral cavity is a compartmentalized, segregated entity unrelated to the gastro-intestinal tract, endocrine system, environment, emotions, etc. Furthermore, if gingivitis and periodontoclasia are only local diseases related to bacteria, local function and local blood flow, they should be simple enough to treat. Use a bactericidal mouth wash, grind teeth so that they do not interfere with each other and keep the patient's heart functioning well (!). All people with malocclusions should then have periodontoclasia. Unfortunately this is not the case. On the other hand, all people who don't perform good mouth hygiene should have periodontoclasia. Unfortunately again many people, primitive and civilized, who never used a tooth brush have no gingival disease. Bacterial accumulations can be "disease producing" only if the local tissues or the patient in general are in a state of lowered resistance. In other words the susceptibility to disease may be of a local *or* a systemic character. Going back to the fundamentals of bacteriology we find that not all oral bacteria are pathogenic; in fact, by far the greatest percentage are nonpathogenic. Therefore, in a state of health the oral flora is harmless and no pathologic conditions occur. However, if tissue resistance becomes lowered, then the bacterial balance in the mouth changes, tissue infection occurs which may sometimes develop rather virulently, as in the ulcerative type of paradental lesions.

In the second paragraph, Dr. Leonard apparently seeks considerable discussion for he corrects his first statement that periodontoclasia is essentially local by stating that some people under unfavorable local conditions do not develop periodontoclasia, and others under more favorable conditions do acquire the disease. This is most certainly indicative of interfering systemic factors, which allow the paradentium to become fertile ground for degenerative processes.

Dr. Leonard reveals his attitude toward systemic conditions, as if they were elementalistic specifics affecting oral tissues, in much the same way as mechanical or physical trauma; whereas the accurate notion of systemic factors or conditions is expressed in the relationship between something happening, let us say in the endocrine environment (*e.g.*, low BMR, hypoinsulinism, etc.), or in the nutritional environment (*e.g.*, B vitamin deficiencies, etc.), or in the emotional environment (*e.g.*, anxiety tension status, etc.), and the subsequent lack of integrity and resorption of the paradental tissues is *manifested by the symptoms* of gingivitis and periodontoclasia. Similar assumptions are used by the great majority of periodontists, namely, that the symptoms of paradental diseases are the "diseases" themselves and that treatment of the symptoms, viz., scaling, surgery, etc., constitute the treatment of the disease.

Dr. Leonard states that the dietary factors about which we have some knowledge can be summarized into three known systemic conditions which lead to "loosening and bone loss prior to gingival inflammation." He names scurvy, osteitis fibrosa cystica, and a

high alkaline diet. According to Dr. Leonard these conditions are the only ones definitely known today. I hope that my statements are not taken to be too critical, but I am embarrassed to say that whenever I cannot find a local factor involved in a case of paradentosis, I certainly would never think of scurvy, osteitis fibrosa cystica or a high alkaline diet. I cannot see why these three diseases have been singled out because there are innumerable endocrine disturbances, nutritional deficiencies, and general systemic disorders that can lead to resorption of bone structure prior to gingival inflammation, resulting in osteodystrophies and osteoporosis. On the other hand, the initial statement leads us to believe that the systemic types of periodontoclasia are those which are characterized by a loss of bone in the absence of inflammation. However, it must be remembered that *endogenous* factors can affect separately gingival tissue without involving primarily the osseous structure and vice versa. And among these endogenous factors nutrition plays a most important role.

Scurvy is certainly not the only vitamin deficiency leading to the characteristic features of the systemic type of periodontoclasia. In clinical paradentic practice, vitamin C deficiencies are found in considerably less than 50% of cases. In our experience the vitamin C in a surprisingly high percentage of cases of pronounced paradentic conditions, is high, and the beneficial effect of vitamin C has been greatly overestimated. Since the studies of Wollbach and Howe, and later those of Boyle, the profession had great hopes that one of the most important etiologic factors of paradentosis had been found. In general, specific results in paradentic practice with vitamin C are very disappointing and the blood tests for vitamin C are not too encouraging either. Scorbutic oral lesions are very rare.

Osteitis fibrosa cystica is not the result of a "dietary factor," but of hyperparathyroidism, i.e., a hormonal disturbance, and is clinically not related to paradentosis. And finally, the term "high alkaline diet" is meaningless and could refer to anything from a high grass diet to a protein-free, grain-free diet. Nutritional records of paradentic patients indicate that a relative preponderance of alkaline-ash foods over acid-ash foods, is comparatively rare, whereas a great majority of diets are preponderantly acid-ash (Walsh).

Vitamin A deficiencies have been grossly overrated as related to paradental conditions, while too little mention is made of the importance of vitamin B complex. Of the various factors of the B complex, thiamin chloride deficiency probably plays the smallest role, while the nicotinic acid avitaminosis or hypovitaminosis takes a more prominent place. Experimental evidence of the importance of nicotinic acid and pantothenic acid for paradental health in animals was presented recently from this department which in the meantime has been confirmed in clinical practice. Large therapeutic dosages of vitamin B complex have proved a great asset in acute and chronic cases of paradental lesions. The vitamin B is water soluble and is lost according to latest investigations (Morgan) to a large extent during the cooking process. Another important point which should not be overlooked is the fact that many of the vitamin B complex fractions are synthesized in the lower intestines by bacteria and that under certain conditions in which general infectious diseases are being combatted by drugs, as for instance, by oral administration of sulfonamides, these beneficial bacteria might be eliminated through these drugs leading possibly to temporary or even prolonged cessation of vitamin B synthesis.

Now as to vitamin D: Does Dr. Leonard really believe that osteomalacia in adults is the result of lack of vitamin D if calcium and phosphates are not abundant and ill-proportioned in the diet? If this were the case, I would think that practically nine out of ten human beings had osteomalacia, because in a recent survey of over 3000 diet records Walsh found that 9 out of 10 individuals were deficient in vitamin D intake. In reality osteomalacia is comparatively rare and, furthermore, neither rickets nor osteomalacia are related to paradentosis. I believe it is generally accepted that vitamin D is not only of utmost importance for the formation of normal bone and teeth, but that its lack may lead to bone resorption and atrophy. We hear so much discussion about the questionable possibility of bone regeneration in paradentics. I hardly believe that vitamin D starved persons have very much of a chance to revive osteoblastic activity and bone formation without optimum administration of vitamin D.

But why is so much emphasis placed on the vitamins? Is it because we are looking

eagerly for that "pill age" when we hope to be able to cure paradentosis solely by tablets and pills? Or is it because we have not paid any attention to the other nutritional factors? Why is no mention made of protein, carbohydrate, and fat metabolism? Furthermore, aren't diabetes and xanthomatosis considered important in paradentics and aren't they related to the nutritional field?

And finally, if the first sentence of Dr. Leonard's last paragraph, "In reviewing this subject, it is apparent that in humans as in animals the first dietary relationship to gingivitis and periodontoclasia lies in the lack of the self-cleansing effect of food," represents in any way the viewpoint of most paradentists, I feel that today in 1944 we have not grasped the importance of modern nutrition to dentistry as yet and are missing one of the most outstanding fundamentals of tissue health and resistance. This statement would imply that if a very quick and hasty analysis of the cause of gingival disease were made, it would primarily be due to "pappy food which fails to cleanse the teeth and massage the gingiva." Could it be that the "pappy food" did not contain essential food factors in form of proteins, minerals and natural vitamins (some of which certainly have not been identified as yet)?

In a recent survey made by Dr. Michael Walsh on the Pacific Coast, the most frequent nutritional disturbances in paradentic patients were found as follows:

1. Insufficient total protein intake.
2. Insufficient animal protein intake.
3. Excessive fat intake; mostly vegetable fats.
4. Excessive intake of carbohydrates derived from grain and refined sugars and starches.
5. Low calcium intake (many as low as 100-200 mg. per day).
6. Low phosphorus intake.
7. Low calcium, high phosphorus ratio, 1:3 or more.
8. Low iron intake.
9. Disturbed acid base balance.
10. Low vitamin B complex intake, especially thiamin, riboflavin and niacin.
11. Very few vitamin C deficiencies.
12. 90% deficient in vitamin D (excluding that from sunshine).

Because of the disturbed Ca/P intake and ratio, low vitamin D intake, and also other factors, many cases show considerable osteoporosis.

The subject of systemic relationship (including the nutritional field) to oral disturbances with special regard to paradental conditions is probably the most neglected field of investigation by clinicians as well as research workers. In spite of the fact that evidence of therapeutic successes by nutritional correction has been coming forth sporadically from dental practitioners as well as through experimental work, it is most disappointing to see that specialists in the field of periodontology who have qualified for certification by the American Board of Periodontology pay little, if any, attention to the fundamentals of nutrition as they apply to modern medical and dental practices and feel that they have reached the highest degree of specialism when they command the digital skill of one or another operative procedure to treat paradental pockets "symptomatically."

I take the liberty of expressing the hope that the Academy as the official organ of the specialists in paradentics will continue to take active steps to rejuvenate the mechanical minds in our midst, and see that they learn the principles of nutrition, and begin to see beyond the bottom of the sulcus tissue reactions on a microscopic level as part of biological processes which are not unique for the oral cavity, but part of the functioning mechanism of the human body.

DICKSON G. BELL, D.D.S.,
San Francisco, California

The published investigations on the systemic aspect of periodontoclasia have shown the broad scope that this phase of the subject embraces. This long list of etiologic and contributing factors emphasizes quite definitely the interrelationship that exists between general body health (function) and the gingival tissues. It is important that we consider the probability that such may exist in all our periodontal cases, and especially with those patients under 30 years of age and having extensive resorption of bone or severe inflammatory changes of the gingiva. In 1941, Becks summarized the data from 31 carefully selected references on the systemic background and stated that, "there is no general uniform cause of paradentosis.

The pathogenesis of paradentosis is different in every case and the disease can develop as the result of certain exogenous conditions as well as internal ones."

In his Initial Statement, Leonard has attempted to interpret the confusing mass of literature on "the nutritional factors relating to gingival and periodontal health and disease." Much of the experimental work has been done on animals. Animal research has its limitations in dental research because it is carried on under most favorable conditions and only the best animals are used. Dentists must treat patients who are in good health and many who are in poor health, as well as those whose psychological difficulties complicate their medical, dental, and nutritional problems.

Radusch (*J.A.D.A.* 28:1524, 1941) discussed this subject of nutrition and periodontal relationship and reviewed the literature of 53 references from which she concluded that,

> From the research reviewed, especially that on mineral metabolism, it would seem reasonable to assume that the tissue response is due at least in part to the nutritive status. Dietary factors can influence the health and disease of the dental tissues, but the present status of knowledge is such that for the most part, answers can not be given as to specific dental application.

Clinical observation of the past few years has shown an increasing number of periodontal patients in the 25 to 35 age group, some even between the ages of 20 and 25. It is interesting to note that a very high percentage of these patients are young women. Men of this age group are usually quite active physically, eat more food, and have a better protein intake, whereas, so many young women are thinking of their waist lines, restricting their food intake or smoking so much that they have no appetite. The result is a mineral and a vitamin deficiency. Roentgenologic examination of most of these cases shows extensive resorption of bone and frequently a decrease in the number of bone trabeculae. Clinically there is a marked inflammation of the gingival tissues with many areas having the deep and vertical type of pocket formation.

In these so-called systemic types of periodontal cases, local therapy has been supplemented with simultaneous treatment or correction of the systemic disturbances. Probably a better plan would be to treat the systemic factors for a month or two before beginning local therapy. Surgeons have long recognized that patients are a better risk when prepared for surgery by administration of a balanced diet for a period of time preceding the operation.

During the past year circumstances required this procedure in several of my periodontal cases. Medical examination had disclosed hypothyroidism and there was hypovitaminosis in each case. These patients were given thyroid and the dietary factors were corrected, including supplementary vitamin B complex. For one reason or another local periodontal treatment of these cases was postponed for several months. At that time it was interesting to note that there was a definite and generalized improvement in the character and appearance of the gingival tissues. The fiery red areas were not so pronounced; the patients reported less bleeding when brushing the teeth and no tenderness of the tissues. In each case, the tissue response to subgingival curettage was more rapid and satisfactory than when treatment followed the orthodox procedure of immediate local therapy with simultaneous correction of the systemic disturbances. This was particularly noticeable in the areas of the deepest pockets, where in these particular types of cases, the tissues usually respond somewhat sluggishly, the redness persists for a while; they require some coaxing and persistent gingival stimulation. In these several patients who had pre-operative systemic treatment, the healing process, tissue tone, color, etc., were comparable to those obtained in the local inflammatory type of periodontoclasia, i.e., a rapid return to physiologic health.

Vitamin D leads to better assimilation and retention of minerals. Diets of periodontal patients should contain not only foods rich in Vitamin C but also an abundance of meat, milk, eggs and cheese, which are not only protein foods but also supply calcium and phosphorus as well as members of the B complex.

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FREDERICK A. BRICKER, D.D.S.,
Los Angeles, California

"Nutrition is the sum total of the metabolic process which maintains the tissue of the individual in a state of health, in contradistinction to diet, which is the food intake of the individual." *(Dr. F. M. Pottenger, Jr.)*

There is an infinite number of degrees of insufficiency and inefficiency resulting from inadequate or unbalanced food intake, inefficient digestion, assimilation, and utilization of food.

Many of us are too prone to think of the oral cavity as a separate entity, failing to consider the body in its entirety. The body is a composite whole. There is a very close relationship between the stomach and the teeth and gums. The gums are not isolated tissues. They are part of the whole wonderful histologic set-up of the body; of the intricate chemical reactions which make a man. Man is a pyramid of elements. When any part of the body is below par, many other parts are affected, and the mouth is almost certain to be one of them. Why else is the tongue one of the first things many physicians examine? I believe the day will come when he will look at the gums with equal care. The gums carry tales that have never been told. I have spent many years teaching my patients how to care for their mouths properly, and emphasizing the importance of the same spotless cleanliness of the mouth as they exercise on their person.

I realized that I was having a certain number of failures, but it took me a long time to discover why. I was not then familiar with the researches of chemists and nutritionists, of men and women like Alexis Carrel, McCollum, Mary Swartz Rose, George W. Crile, and Gotting and Galan.

We must take the lead in emphasizing the relation between sound body tissues and sound teeth and gums. It is very definitely our responsibility. How many of us can conscientiously say that when he is confronted with dental periclasia and dental caries in the mouth of a patient whose oral hygiene is meticulous, he immediately suspects malnutrition or refers the patient to his physician to find out if there is not some systemic disease influencing the mouth condition?

We cannot practice periodontia creditably until we ourselves make a serious study of the chemistry of nutrition, and the science of histology; nor can we build up coöperation with a large number of physicians until we learn the art of diplomacy. A closer relationship between dentistry and medicine is daily becoming more apparent.

Modern studies of the etiology of dental caries and dental periclasia are based upon the assumption that the health of the body as a whole determines the immunity or susceptibility to these diseases and most other diseases.

The biochemical aspect as related to periodontal tissues is very important, and when one refers to tissue reactions, the term indicates two relationships; (1) fluctuation in the composition of the tissue cell, and (2) the relative changes which take place between the cells in the inter-cellular substance and the surrounding fluids.

If dental periclasia exists, one will find that one or both of these relationships has been altered. No tissue is more prone to show inflammatory reaction than the delicate gingival tissue. Involvement of the marginal gingiva tends to spread to the cemental and alveolar portions, with varying degrees of severity, depending to a great extent on the type of bacterial invasion and its toxins, but in direct ratio to the tissue's resistance.

By tissue resistance, is meant the ability of living tissue to remain healthy in the face of adversity. Living tissue, which is normal, protects itself efficiently against injury, whether that injury be mechanical, chemical, or bacterial.

Various authors have helped us to understand the chemistry of tissue and its relationship to nutrition.

Mary Swartz Rose writes:

Mineral elements are essential to the structure of certain complex chemical compounds which profoundly influence the course of metabolism. The body is constantly losing some of each mineral element. It becomes necessary to replace the loss through diet or health will not be maintained. These must be nicely balanced in order that all parts of the body may function successfully.

McCollum has shown that, "No less than 37 minerals are essential for an adequate diet, 18 amino acids, 1 glucose, 11 mineral salts, 1 fatty acid and 6 vitamins. Of these, minerals and vitamins are deemed of special importance to dental health."

In the 1939 *Year Book of Agriculture*, we find this statement under the subtitle Food & Life.

Certain Minerals and other inorganic substances are commonly called trace elements because they are found, in the body or in food, in very minute amounts, mere traces. With the ordinary chemical methods of analysis, the presence of many of them was never suspected. They were revealed by the spectrograph, an instrument used to show what the stars contain and to detect slight impurities in metals. Then, it turned out that some of these trace elements in soils, in plants, in animal bodies,

and in the human body, play an extremely important part in life processes.

Organic defects are not the only ones which influence physical well being and through it, the gingival tissue. Dr. George W. Crile, the famous Cleveland physician, has again and again emphasized the importance of emotional factors, such as worry and fear. In *Phenomena of Life*, published in 1936, he wrote:

Worry is a state of alternation between hope and fear. It is an alternating stimulation and depression of oxidation, radiation, and electric potential. It is a slow fading out of the molecular furnaces within the protoplasm. In accordance with this conception, we can understand why the man consumed with financial (or family worries) suffers so many bodily impairments; even diseases. We can understand the grave digestive and metabolic disturbance which appears under any strain of worry or fear. When an individual is subjected to emotion, all his protoplasm is affected.

We quote from Mary Swartz Rose, *Foundations of Nutrition* (1938): "Sometimes the root of undernutrition is worry, unhappiness, fatigue, a veritable destroyer of appetite."

Dr. Alexis Carrel, in *Man the Unknown*, published in 1935, writes in a similar vein.

Thought can generate organic lesions. The instability of modern life, the ceaseless agitation, and the lack of security, create states of consciousness which bring about nervous and organic disorders of the stomach and of the intestines, defective nutrition, and passage of intestinal microbes into the circulatory apparatus. Colitis and the accompanying infections of the kidneys and of the bladder are the remote results of mental and moral imbalance.

All systemic and emotional changes are basic factors in periodontal disturbances. They interfere with nutritional metabolism and should be dealt with as primary factors.

There are of course local secondary factors such as are mentioned in the initial statement.

Nutrition is not a simple science. It can be successfully taught only by the same careful selection and adaptation of subject matter which characterizes the successful teaching of other subjects.

Man's body, like an engine, requires fuel, or energy, which must be derived from his food. The essential elements of food which furnish energy and heat are carbohydrates, proteins and fats. These food factors have been given great attention in the past, but vitamins, mineral salts, and water are also indispensable for the maintenance of normal nutrition and are especially important to dental health.

Appetite is seldom trustworthy. How much and what one eats depends largely upon taste and the attractiveness of the food. Appetite rarely uses good judgement in the selection of proper foods.

In order to make nutritional corrections intelligently, we need to know as much as possible about the various mineral elements required. For example, some elements such as calcium and phosphorus are required in relatively large amounts, and the intake balance is extremely important, and because of this—there is a possibility of a serious deficiency, unless the food is chosen with discrimination.

A survey made by members of the American Academy of Applied Nutrition, a group composed of dentists and physicians, organized eleven years ago, who meet monthly to study and discuss nutritional problems in their individual practices, report these findings:

In 3000 diets, records of every 10 diet analyses, these dietary deficiencies occur:

6 out of 10 were deficient in vitamin A intake
9 out of 10 were deficient in vitamin B intake
2 out of 10 were deficient in vitamin C intake
9 out of 10 were deficient in vitamin D intake
4 out of 10 were deficient in vitamin B_2 (G) intake

6 out of 10 were deficient in total energy food intake
8 out of 10 were deficient in total protein intake
7 out of 10 were deficient in total calcium intake
7 out of 10 were deficient in total phosphorus intake
8 out of 10 were deficient in total iron intake
9 out of 10 were deficient in total iodine intake

From my own case histories over the past 15 years, the most common nutritional defects noted in dental periclasia are nutritional anemia, nutritional deficiencies, mineral imbalances, *high* fat and carbohydrate intake, and *low* milk, cheese, eggs, meat, vegetables, and fruits. A *low* nutritional level.

Let us as dentists, especially those of us

who have accepted the title of specialist, acquire an adequate knowledge of nutrition, so as to be qualified to evaluate diets and to direct our patients in the selection of an optimum, well balanced diet containing an adequate number of calories, fats, carbohydrates, proteins, and the necessary vitamins, water, and minerals.

As I see it, that bright glaring electric sign advising the teaching of mouth brushing to prevent dental caries and dental periclasia is being outshone by a brilliant fluorescent sign stressing the importance of building up and stimulating the natural defense mechanisms of the body as a whole in order to prevent these diseases.

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It seems to me that Dr. Leonard's Initial Statement is well calculated to accentuate the trifling amount of real accomplishment and the chaos of opinion on the etiological factors of periodontoclasia. So long as we do not positively know, we may as well opine that this, that, or the other factor or set of factors, or all of them acting concurrently, independently, or even consecutively, may account for the regrettable loss of teeth. Certainly, nutritional factors should be entertained as among those most likely to constitute an etiological entity, or to be contributory if not initiatory in the sequence of events.

Whether we like it or not, the slogan "we ARE what we eat" is an applicable truism. However, it is only a half-truth and as such, might mislead one into placing the emphasis on *what* we eat instead of upon the disposition of it. Starvation may be accomplished as positively by failure to utilize as by failure to ingest essential nutritional factors. The fact that Americans as a whole eat well and yet exhibit relatively as much periodontoclasia as other nationalities does not disprove or dissociate nutritional factors in this connection. By the term "nutritional factors" is meant here everything relative to metabolism. It is a matter of common observation that victims in famine-ridden lands exhibit certain signs so regularly that they are characterized as symptoms of malnutrition. There is in such instances, no question of food substances or assimilation being the etiological factor, but the part played by avitaminosis is problematical since there is evidence that some of the vitamins are auto-synthetisized, and these presumably being present in famished individuals can be indicated only as accessory to the fact. On the other hand, individuals, regardless of geographic distribution who command and consume a complete assortment and variety of food stuffs, exhibit signs of malnutrition. It is not unreasonable to assume that an occasional individual who exhibits undernourishment is unfortunate in both procurement and assimilation. Seemingly, periodontologists are in agreement in their interpretation of the comparative radiolucent areas about the roots of teeth. Unquestionably the integrity of the bone is being disrupted, and the degree of destruction is roughly proportional to the radiolucency. We are quite in accord on the necessity of removing all contributory sources of irritation although we deviate somewhat on the manner of procedure. We further agree that some cases exhibit terrific bony destruction in almost complete absence of gingivitis, the original cervical gingival attachment being intact. Acceptance of a premise of "an inward reaction" in such cases seemingly is inescapable, and we are obliged to attribute such premise to our present-time limited assortment of etiological factors—lack of food; inability to assimilate food; endocrine imbalance which presumably may interfere with proper distribution of food.

Our success with these cases at Iowa State University probably approximates the average results over the country. For several years we have been advising our patients on the importance of an inclusive dietary, emphasizing the necessity of intake of certain elements in sufficient quantities and the intake of the agents or vitamins responsible for their utilization. It has seemed to us sufficient to impress the suggestion of a wide variety of all kinds of foods in digestible amounts rather than attempt ourselves an analysis in an unfamiliar field. Where this fails to improve the general health of the individual, we feel that it is unlikely that the local condition has benefitted and we advise securing the services of the hospital staff who are equipped to investigate and interpret their findings. In the meantime, we proceed with the standard practice of removal of irritation and establishment of oral hygiene, the while insisting upon general health building measures.

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The prevention or treatment of gingivitis and periodontoclasia would be simple if the administration of vitamins were the only requirement. It is true that scurvy is treated and cured by the administration of Vitamin C and that pellagra responds to niacin and a well balanced diet. Manifestations of thiamin and riboflavin deficiencies may be cleared quickly by the use of therapeutic doses of these vitamins. The tendency is to conclude that because gingival disturbances may be evident in some avitaminoses that gingivitis and periodontoclasia are also manifestations of an avitaminosis.

Those who manufacture or sell vitamins are reaping a harvest by the sale of their products. Almost all radio programs are sponsored either by soap or vitamin manufacturers so that the laity is constantly bombarded with pleas or threats to purchase and take vitamins. Consequently, too little attention is given to the selection and consumption of foods which contain vitamins and other important food elements known to be needed for human nutrition.

The relation of vitamins to gingivitis and periodontoclasia is as yet not clear. In the case of Vitamin C there are those who assert that there is no difference in the ascorbic level of those who have gingivitis and those who have normal gingival tissues. There is a considerable number of investigators who take the opposite view. Much more research is necessary and much more must be known about vitamins before the problem is settled.

There is a tendency now to claim that adequate amounts of vitamins will cure gingivitis and periodontal disease just as several years ago vitamins were going to save humanity from dental caries. Now it is becoming increasingly clear and widely accepted that teeth may be "fed" with all the vitamins known and they may continue to decay if sugar is included in fairly liberal amounts.

It is not our intention to minimize the value or importance of vitamins, but we have sent many patients in from the Dental Clinic and our practices with gingival disturbances for thorough examination at the hospital, expecting to receive a report of definite systemic findings and have been told that there are no significant findings.

I am not clear as to an assertion near the conclusion of the Initial Statement,—a very fine resumé of this matter of the relation of nutrition and gingival disturbances. It states that, "excessive consumption of sugar . . . acts not only to increase caries but also to increase bacterial growth which *causes* gingivitis." There is at present no evidence that the organisms which produce caries cause gingivitis. In fact, the evidence is rather to the contrary. Gingivitis is more probably associated with proteolytic organisms than with aciduric organisms.

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Nutrition is the sum total of the processes by which growth is promoted and health maintained. The word is too often confused as a synonym for diet, which itself is a part, but only a part, of nutrition, as are digestion, absorption, utilization, and excretion. All factors affecting any of these processes in the cell, the organ, or the organism must be considered if we discuss nutritional factors in any disease.

Texts in medicine, dentistry, and surgery are concerned with disease entities, often with a disregard for the rest of the organism, which is possible only on the dissecting table. This compartmental thinking, together with increased specialization, has retarded advances in our understanding of what constitutes health. Not infrequently, we hear a mother state that the pediatrician says her child is in perfect health, yet both agree that the child needs orthodontic treatment, has some carious teeth, and as an afterthought, is "allergic." It is neither logical nor possible to believe that a disease process may occur in any part of a living organism without a departure from the normal in other parts.

Essentially, gingivitis and periodontoclasia are local manifestations of systemic abnormality, usually exacerbated by local factors in the early stages, invariably so in the more advanced stages. The second paragraph in the "Initial Statement" well illustrates the point. With an entirely normal systemic state, local factors cause no apparent lesions. If a systemic abnormality of relatively small degree exists, then the removal of all local factors enables the tissues to repair, through their own vital resistance, and the pathologic disturbances are arrested. Where the systemic abnormality is serious, then local treatment seems of little or no avail.

In the cases of gingivitis and periodonto-

clasia I have observed, nutritional deficiencies have been demonstrable. Usually, these have been dietary, frequently complicated by metabolic inadequacies, but in a very few cases where dietary deficiency was known to have been obviated for one to three years, minor endocrine insufficiency was the sole deficiency, with the exception of certain diabetic cases.

A good diet is one which supplies the individual with all the elements for growth and health under his particular environmental conditions. No general statement can be more specific. The primitive Eskimo maintains health on a diet entirely different from that of the Arab, whose diet nevertheless has maintained him in a similar state through countless generations.

The British medical officer, McCarrison, after observing for years the superb development and health of the Hunza people of the Himalayas, reported, in 1907, the results of extensive experiments with the Hunza diet. He found that rats fed the total diet reproduced normally and maintained health generation after generation, but the omission of any part of the diet resulted in rapid decline.

In animal feeding experiments, certain deficiencies are imposed or restored with observed results, but never is a simple specific lesion produced, such as periodontoclasia, for example, without other changes which may have to do with the hair, skin, intestinal tract or otherwise. Animals fed by man show periodontoclasia through failure to receive all the elements needed. Wild animals incapacitated through age or injury or failure of supply may show the same. Dr. Becks has reported the effects of withholding certain fractions of the B-complex from dogs. Dr. Pottenger has demonstrated extensively the effects of loss of factors destroyed by heat in the feeding of cats. Periodontoclasia ensued, but no essential difference in the coarseness of the foods existed. Admittedly, exercise of the part is beneficial, but we are prone to adopt half-truths as explanations, as for example, attributing the increase in orthodontics to more efficient recognition of the needs. Whereas, in fact, we fail to recognize the greatly increased need among children of parents with normal jaw development.

Dietary calculations for an individual must take into account not only the factors of age, weight, activity, environment, etc., but also previous environment, heredity and racial or geographic conditioning. This is illustrated in people of Scandinavian extraction, who racially have been conditioned to a high mineral intake through the large use of sea foods, and who show a low survival value in inland areas.

It is entirely logical to expect that a highly vascular bone such as the alveolar process and the ribs will show greater resorption and decalcification under the influence of demineralizing systemic conditions than will a less vascular bone. Furthermore, it is important to remember the pathologic fact that inflammation which in soft tissue produces proliferation, in proximity to bone causes resorption. Thus, any condition which produces inflammatory soft tissue (gingivitis) will ultimately cause bone resorption (periodontoclasia). In the absence of local irritants or inflammation, this same high vascularity of alveolar process acts as a protective measure.

It is my understanding that the figures published by the Food and Nutrition Board of the National Research Council represent minimal maintenance requirements, and that those given for protein are for total protein. This latter is supported by the suggestion that certain vegetable proteins be substituted in part for high grade animal protein. Certainly this figure for protein requirement is far from optimal in practical application. It is of interest to note that analysis of the diet furnished for the rigors of the St. Mary's Pre-Flight School in California allowed 325 grams of protein daily, the bulk of it high-grade animal protein. This is evident recognition of the fact that only from protein is new growth and repair of tissue effected.

In reviewing this subject, it is apparent that in humans and in animals the chief dietary relation to gingivitis and periodontoclasia lies in the lack of essential elements, minerals, vitamins, and other protective factors, rather than that of mechanical cleansing effects. In the nutritional approach to these problems, it is important to realize that the lesions discussed are the visible evidences of a disturbed metabolism. While recognizing the specific demonstrable effects resulting from the omission of certain vitamins or other dietary factors, we know that under present living conditions, particularly in cities, neither causes nor results are ever single.

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The "Initial Statement" says that "Essentially, gingivitis and periodontoclasia are local diseases related to bacterial accumulations, local function, and blood flow. Systemic conditions modify this process in many ways, some of which are known and many as yet unknown." This premise is open to question. According to the scientific data now available, it would be just as proper to say that periodontoclasia is a systemic condition which is modified by local factors. This premise would encompass all types of cases, including those exceptions to the "local disease theory" mentioned by Dr. Leonard when he says "Every periodontist has seen cases in which there was every local reason for gingivitis and periodontoclasia to be present, yet the gingival tissues were healthy. Also he has seen cases in which the most exacting local treatment and home care brought health only very slowly or incompletely."

Unfortunately the "local theory" has been commonly accepted, because many cases apparently respond favorably to local treatment; but, such response is no proof of primary local causation. A great number of disease conditions respond favorably to local treatment; for example, tuberculosis. Yet, no one would state that local factors were the cause of the disease.

Thus, even though it may be believed that the fundamental factors in periodontal disease are systemic, that does not prevent the use of local measures in treatment in so far as possible. Scientific knowledge is yet of such a state that it does not permit ready diagnosis of constitutional factors, unless they are gross.

There is considerable published data indicating the importance of the systemic condition. Referring to the role of nutrition, the "Initial Statement" specifies scurvy, osteitis, fibrosa cystica, and a highly alkaline diet as factors leading to loosening of teeth and bone loss prior to gingival inflammation. But there are many other phases to be considered.

There is a good deal of research which points to the importance of calcium intake. A recent report is that of Lund and Armstrong *(Proc. Soc. Exp. Biol. and Med.* 50:363, 1942) in which, when rats were kept for five weeks on a low calcium and phosphorus intake, alveolar bone was found to be soft and friable, the alveolar crest resorbed and teeth loosened; while in the control rats, the crest was normal and teeth were firmly fixed. Anderson and associates *(Yale J. Biol. and Med.* 9:189, Dec. 1936) said that bone changes and shifting of teeth in rats were greater in those kept on low-mineral diets than in those on adequate diets. Changes produced by the local factor of occlusal stresses, were definitely modified by the nutritional status of the bone. Since these animals are not in need of dietary vitamin C, the work is especially significant. It must be remembered, too, that the highly alkaline diets which have been projected as being disadvantageous were, for the most part, calcium low and often low in other factors as well.

The gingival tissues are particularly dependent on the vitamins for their health status. Research by Topping, Fraser and Tomlinson *(Pub. Health Rep.* 54: Mar. 17, 1939) on monkeys "fed on diets deficient in certain vitamins resulted in the development of necrosis of the periodontal tissues and oral mucosa of varying extent, qualitatively similar in the B_2 complex deficiencies with variations in the flavin and nicotinic factors, in the C deficiency and in the D deficiency, and probably greatest in extent in the B_2 deficient groups." They found little or no change clinically or histologically in the case of exceedingly low vitamin A intakes. Numerous pertinent studies could be cited, but space does not permit.

In the concluding paragraph, the "Initial Statement" says, "It is apparent that in humans as in animals, the chief dietary relation to gingivitis and periodontoclasia lies in the lack of self-cleansing effects of food." How can this be apparent, when there is a paucity of data relating to local action of food?

May I refer to the discussion of this subject in the *J.A.D.A.* 28:1529, Sept. 1941. In brief, experiments by Simonton and associates, and Mellanby indicate that balanced diets resulted in cleaner, healthier mouths than even detergent diets. Although Burwasser and Hill, after experimenting on five dogs stated that soft diets tend to cause more pathologic changes of the gingivae, the small number of animals used precluded any positive conclusions. Primitive races, in spite of diets requiring function, are reported as usually suffering from gingival diseases.

Lastly, it seems quite premature to make the prediction in the following statements: "As regards primary underlying connective tissue changes, severe scurvy, alkaline medication, and hyper-parathyroidism are known at present. Others remain to be discovered but will probably be found to be hormonal or toxic conditions and not nutritional excesses or deficiencies." Research along these lines is only barely started, but, even so, that already done on vitamin and mineral deficiencies is highly indicative of the importance of nutritional factors. More particularly so, because studies show clearly that dietary intake alone of essential nutrients is commonly inadequate. When this is considered in conjunction with utilization, the other phase of nutrition, one can safely say that nutrition plays one of the fundamental roles in periodontal status; probably both directly as it affects the structure of the periodontium and indirectly as it affects general health and resistance.

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The topic for discussion, "Nutritional Factors Relating to Gingival and Periodontal Health and Disease," involves one of the most fascinating chapters in the history of the practice of medicine. Results being obtained from the application of the important science of nutrition in the health and welfare of the Allied Armed Forces, promises to be one of the significant accomplishments of modern medical science.

It is interesting to note that Kruse (1) states that,

> Deficiency diseases have been shown to be widespread in previously unrecognized forms: mild acute, mild chronic, and severe chronic. The acute and chronic processes may be present together. Indeed, not infrequently an acute is superimposed on a chronic condition. Each may be in a different stage and of a different intensity. It should be emphasized that this refers to each kind of deficiency disease. Characteristic tissue changes reflecting form, degree, and stage have been demonstrated in each of four deficiency states. So common, so prevalent, so frequently seen are some of these changes, especially their slighter degrees, that they have been regarded as usual or normal. But their specific location, distinctive and characteristic nature, their reversibility, all attest to their pathological identity as deficiency states . . . the prevalence and severity of the chronic processes increase with age. Most of the chronic changes have hitherto been regarded mistakenly as characteristics of senescence. In reality, however, they are deficiencies developing slowly over a lifetime. These chronic processes may be seen beginning in childhood, even in infancy. The lower the economic level, the more frequently, the more severe, and the earlier in life they occur. A person's nutritional status today depends on the events of all past days. Upon reflection it is clear that few people have had a satisfactory intake or escaped adverse conditioning factors every day of their lives. These lapses are reflected in tissue changes, and, until recently, attempts were seldom made to correct them entirely. Indeed, many persons continue on an inadequate diet for years. Hence, the tissue shows the cumulative changes of long, slowly developing chronic as well as rapidly progressing acute processes. All points considered, it is to be expected that the prevalence of deficiency states is high.

Vitamins, whether they be enzymes, coenzymes, activators or exogenous hormones, are necessary for the endogenous metabolism of tissue proteins, carbohydrates, fats, and minerals. The qualitative rather than the quantitative aspect of the dietary problem is of great importance. A variety of proteins is also required in order to maintain the essential amino acids required for proper nitrogen equilibrium and cellular metabolism. It should be fully realized that as vitamins occur together in nature, and factors that influence absorption, utilization and increased requirement affects not one but many vitamins, deficiency diseases are apt to occur as complexities rather than as single entities.

As the mouth is the outstanding mirror of pathology those diseases so reflected are usually involved in gingival and periodontal diseases. Some of these conditions may be difficult or impossible to control, they are usually clinical reflections of severe or dangerous diseases. A slow or non-response to treatment may indicate such a condition. When a diffuse inflammation extends beyond the gingiva, or includes the buccal mucosa, tongue or lips, with a hemmorrhagic tendency, a rare or serious causative factor must be looked for. Local treatment usually exaggerates, rather than helps the condition.

Animal experimentation by leading investigators in the field of nutrition have added greatly to our knowledge of deficiency diseases as it pertains to dental involvements. Boyle, Bessey, and Wolbach (2) were the first to call attention to the similarity between diffuse atrophy of the alveolar bone type of pyorrhea and the tissue changes in vitamin C deficiency. Becks and Morgan (3) presented the experimental evidence of a relationship between vitamin B fractions and dental and paradental dis-

orders. They conclude that, "the filtrate fraction of the B complex and nicotinic acid must be recognized as essential food accessories for the health of dental gingival and paradental structures because their absence leads to severe gingival inflammation and infection of the underlying osseous structure. Nicotinic acid deficiency alone appears to lead to gingival disturbances."

Topping and Fraser (4) of the United States Public Health Service conducted vitamin deficiency experiments on monkeys. Observations presented indicate that certain of the dietary deficiencies tested (Vitamins A, C, nicotinic acid, flavin and nicotinic acid and B_2 complex control diet, containing flavin and nicotinic acid) are associated with the development of varying manifestations of gingivitis, stomatitis, periodontal disease, and noma. Animals maintained on adequate diets showed little or no evidence of such oral pathology. It is interesting to note that these authors, in order to test infectivity and virulence of the spirals, fusiform, and other organisms occurring in the mouths of those monkeys developing lesions, made several direct transfers of the contaminated material to the mouths of normal monkeys. The gums of the normal monkeys were lacerated; teeth were extracted and the material swabbed into the areas. No gingivitis or stomatitis resulted from this procedure.

It seems reasonable to assume that those factors that retard healing and interfere with healing of wounds in general may be involved in gingival and periodontal conditions. Factors that delay or inhibit wound healing certainly influence the response to local treatment. Perhaps the most common or widespread conditions are hypoproteinemia and vitamin C deficiency. Lund and Crandon (5) in their discussion "Nutrition as it Affects Wound Healing" seem to cover these assumptions. They state,

> The symptoms, signs, and gross pathology of scurvy have been long understood. However, the fact that intercellular collagen could not be formed in the scorbutic state is comparatively recent knowledge. Although ascorbic acid has an important intercellular function as an enzyme effective in oxidation-reduction systems, it is the failure of deposition of collagen in its absence that causes failure of healing . . . In 1930, it was shown positively that the protein intake of dogs was related to the rapidity of healing of wounds, but it was not until 1938 that Thompson, Ravdin and Frank showed that demonstrable hypoproteinemia in man is associated with delayed healing in wounds. It is particularly interesting that the degree of hypoproteinemia does not have to be great to cause this effect. A reduction of 15% in plasma protein may lead to moderate delay, while a reduction of 25% is severe enough to cause very serious delay or complete failure of wound healing . . . Dietary correction of hypoproteinemia is sometimes very slow and sometimes impossible.

Smith (6) has stated that, "Vincent's infection is a common secondary infection in vitamin A, C and the pellagra deficiencies. It also occurs when there is radium poisoning, benzene poisoning or leukemia." Our experience indicates a lack of specific factors responsible for acute Vincent's infection. Whereas nicotinic acid deficiency seems to be involved in many cases, we have had cases that did not respond to massive doses (600 to 800 milligrams daily). This may also be said of vitamin C deficiency. Failure of response to 600 to 1200 milligrams daily occurs in many cases. Failure of vitamin therapy alone may be expected in chronic alcoholics, where the caloric intake is restricted to carbohydrates. Drug addicts present a similar condition. Also in patients recovering from a prolonged illness, where protein intake has been limited.

Boyd's (7) description of the oral manifestations of vitamin C deficiency is very similar to that of diffuse atrophy of the alveolar bone. He states that "the gums are soft, spongy and bleed readily and the teeth may fall out, bone formation is brought to a standstill, and as the normal process of bone absorption still goes on the bones become rarefied and fragile."

The clinical manifestations and pathologic lesions of diffuse atrophy of the alveolar bone, as described by Kronfeld (8), resemble closely the scorbutic lesions of vitamin C deficiency as above described. This author states: "Loosening and pathological wandering of the teeth, widening of the periodontal space with transformation of fibrous periodontal membrane into loose vascular connective tissues, rarefaction and extensive loss of alveolar bone are the outstanding features."

As "the tendency in vitamin C deficiency is for defective material to form in connective tissues in partial depletion and for nothing of a substantial nature to form in complete depletion" (9) the periodontal metamorphotic changes described by Kronfeld in diffuse atrophy of the alveolar bone are well understood. It is reasonable to assume, from the similarity of the tissue changes in

the two conditions, that the loss of collagenous periodontal fibers may be the important and primary factor in this type of pyorrhea. This similarity is well understood because the periodontal fibers, according to Noyes, Schour, and Noyes (10) are composed of collagenous white fibers. Resorption of these fibers in vitamin C deficiency would naturally weaken the stabilizing mechanism to an extent that the physiological stress of mastication becomes pathological, resulting in the "pathological wandering of the teeth" described in this type of pyorrhea.

Orban and Weinmann's (11) summary of the "Diffuse Atrophy of the Alveolar Bone (Periodontosis)" resembles closely the description of the clinical manifestations of vitamin C deficiency.

In a paper on vitamin A and B deficiency read before the Periodontal Section of the American Dental Association in 1938, Sinclair (12) says: "The presence of chronic Vincent's infection and the slow response or non-response to orthodox treatment of pyorrhea lesions are highly suggestive of a deficiency of one or more vitamins.

"My experience strongly suggests that a multiple deficiency is responsible for the conditions described by Gottlieb as 'diffuse atrophy of the alveolar bone'; by Box, as 'complex periodontitis,' and by Becks, as 'genuine paradentitis.'" *(See* also (13).)

It seems more than probable that avitaminosis and hypoproteinemia may be involved as the important etiological factor, not only in diffuse atrophy of the alveolar bone type, but in the great majority of periodontal diseases, the severity of the lesion depending upon the duration and extent of deficiency. Diffuse atrophy is perhaps the end result of a prolonged severe multiple deficiency, and deficiencies, slight or complete, may be, with few exceptions, the primary etiological factor in all periodontal involvements, with foreign irritants acting only as precipitating factors.

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The initial statement is so comprehensive as to offer only one difference and that is, regarding dilantin sodium. This drug will not produce gingival hypertrophy where there are no accumulations upon the teeth and where strict hygiene obtains. It may be observed that in those patients receiving dilantin sodium who have severe mal-occlusion, the hypertrophy occurs only about those teeth which are not in function. Therefore, rigid cleanliness plus careful massage and stimulation of the gingival tissue will reduce or prevent hypertrophy in those patients receiving dilantin sodium.

It is difficult to write generally of nutritional factors influencing gingival health

without mentioning the prevalence of smoking. Smoking is so common a habit that he who dares lift his voice in criticism of it is courageous indeed.

Nevertheless, the habit is deliberately fostered today by young women for the purpose of stifling appetite and remaining thin. These young women starve themselves by denying their bodies the necessary elements of nutrition and so gingival disorders are seen in their mouths at a distressingly early age.

The psychological factors influencing young men to smoke are undoubtedly not the same. However, by dulling the normal instinct for essential food elements which the habit of smoking produces, a state of inadequate nourishment persists.

A planned educational program for the reduction of smoking and the introduction of a completely adequate diet would be of value in reducing gingival disorders.

DANIEL E. ZISKIN, D.D.S.,
New York, N.Y.

While the subject under discussion is restricted to nutritional factors relating to gingival and periodontal health and diseases, such restriction is not valid in the strict sense of the term. It has been established that gross nutritional disturbances are usually complicated by metabolic disorders. There may likewise be emotional involvements. The converse may also be true that in deep-seated emotional or endocrinological imbalances the body foods no longer function normally.

Nutritional deficiencies are often seen in diseases of various endocrine glands. In diabetes, vitamin A and B complex deficiencies are common. In cases of hypothyroidism there is usually vitamin A deficiency. Vitamin B complex deficiency is frequently accompanied by an increase in endogenous estrogens. Relationships have been demonstrated between vitamin A and estrogen as well as vitamin C and progesterone, and between vitamin D and the parathyroid secretion.

Inanition in rats has been shown to produce symptoms resembling hypophysectomy. Examples of local effects of nutritional disturbances on gingival tissues are ample in the original statement. Examples of the effects of metabolic disorders can also be cited. We have produced Vincent's infection in castrated female monkeys by parenteral injection of prolan. We have reported similar results with prolan injected in humans. Here the clinical symptoms of acute Vincent's infection disappeared upon withdrawal of the drug without local treatment.

In rats on deficiency diets we have observed accumulations of sordes on the incisors which, when examined microscopically, showed the presence of bacteria and tissue debris, as well as abnormal shedding or desquamation of epithelial cells. These tissue elements together with bacteria remain to act as gingival irritants.

Consideration of therapy for gingival and periodontal diseases and evaluation of the importance of systemic therapy as compared with local periodontal treatment, must take into account possible etiology. Gingival symptoms may arise from frank nutritional or endocrine gland deficiencies, such as scurvy. These cases are rare. Their response to substitution is of little value. On the other hand, when doses of vitamins or hormones are given in amounts greatly in excess of physiological requirements these agents can no longer be considered as substitute biological foods but must be regarded as potent drugs. Studies have recently been conducted in which some diseases of the gingivae were found to respond to this form of massive medication. But these results must be regarded in an experimental light until much more data is acquired. Until such time or until other systemic agents become available, we must continue to use established local measures in treating the ordinary case of periodontoclasia, supplemented in certain instances by systemic formulae. In other words, all periodontal disorders cannot be effectively eliminated by vitamin pills or other internal medications. In my opinion, most cases of periodontoclasia can at present be best handled by local means, the intelligent use of systemic measures constituting a valuable adjunct.

Concluding Statement

I am extremely grateful for the critical discussions on this subject especially those of Radusch and Becks. These discussions contribute new knowledge and also a viewpoint which periodontists will certainly wish to adopt as means become available for implementing it.

It is apparent from the discussion that the emphasis in the initial statement on local factors with occasional systemic complications may have to be reversed to considering gingivitis, periodontoclasia, and Vincent's infection essentially as manifestations of impaired systemic chemistry in which local irritants act merely as precipitating agents. We have hitherto placed the emphasis on local factors because we have been able to treat them and by such treatment, with few exceptions, to restore the gingival tissues to health. Until now we have been unable except in extreme cases to determine the underlying chemical fault or to correct it. So we have discounted systemic and nutritional factors as still in the realm of theory.

We now have the means available for estimating nutritional deficiencies in the diet with precision. This should open the way for a new attack on our periodontal problems. A book by Clara Taylor, *Food Values in Shares and Weights*, MacMillan, 1942, makes it a fairly simple matter to estimate a day or week's diet in regard to every nutrient with which we are concerned except phosphorus, niacin, and vitamin D. The book by Frances Stern, *Applied Dietetics*, 1943, includes phosphorus, niacin, and vitamin D figures which may thus be added with some effort. By means of a simplifying device which Professor Taylor uses, a day's dietary can be accurately estimated in half an hour and a week's dietary in an hour.

As the new knowledge of nutrition becomes disseminated and its significance in medical and dental practice recognized, we may expect accurate blood tests for mild vitamin deficiency states to be developed and to become commercially available. Chemical blood tests are necessary because individuals may be deficient even though the diet is adequate according to standards, because vitamins may be lost in the preparation of food or the individual may not be absorbing or utilizing them or may need an excess.

This discussion has certainly been helpful to me as opening a vast field of usefulness to the dentist which has up to now been seen as a mirage with all a mirage's disappointments. With positive tests for dietary deficiencies available, with mouth signs of particular deficiencies becoming better defined and adequate food sources of the different vitamins known, it is possible and practical for the dentist to become a nutritionist. Since dental caries susceptibility probably also lies in this field, it may be possible to inaugurate the era of real preventive dentistry through this means.

HAROLD J. LEONARD

"Good nutrition comes from a wide choice and a wide variety of wholesome food, not from a poor diet supplemented with vitamin and mineral preparations."—FREDERICK J. STARE, M.D.

"VINCENT'S INFECTION"—Subject for discussion in July, 1944 issue.

FOURTH

Postgraduate Program for

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GRADUATE AND POSTGRADUATE DENTISTRY

University of Michigan, Ann Arbor, Michigan

APRIL 24, 25, AND 26, 1944

MONDAY, APRIL 24th

9:00
Registration and Announcements

10:30
Dynamics of Inflammation, Valy Menkin, B.S., M.A., M.D. Formerly of Harvard University.

2:00
Pathologic Circulatory Physiology (Motion Pictures Taken Through a Microscope), Melvin H. Knisely, M.D., Department of Anatomy, University of Chicago.

3:30
Dynamics of Inflammation, (continued by Dr. Valy Menkin).

6:45
Dinner and Speaker: Dentistry in the Navy, Lieut. Commander Harold E. Kerr, U. S. Naval Training Station, Great Lakes, Illinois.

TUESDAY, APRIL 25th

9:00
Pharmacology of the Sulfonamides and Their Toxic Effects, Maurice Seevers, Ph.D., M.D., Prof. Pharmacology, Chairman of Dept., University of Michigan.

10:30
Gingival Inflammation, Balint Orban, M.D., D.D.S., Professor Pathology, Loyola University, Chicago.

1:30
Clinical Application of Sulpha Drugs and Penicillin, H. M. Pollard, M.S., M.D., Professor Internal Medicine, University of Michigan.

3:30
Dermatological Manifestations—Clinical Demonstration, A. C. Curtis, M.D., Professor of Dermatology, University of Michigan.

7:30
University of Michigan Research on Dental Caries, Phillip Jay, D.D.S., D.Sc., School of Dentistry, University of Michigan.

WEDNESDAY, APRIL 26th

9:00
A Classification and Etiology of Periodontal Diseases, Maynard K. Hine, D.D.S., University of Illinois, Chicago, Ill.

10:30
Recent Advances in Chemotherapy, Maurice Seevers, Ph.D., M.D., Prof. Pharmacology, University of Michigan.

1:30
Fatigue. Carl D. Camp, M.D., University of Michigan.

ACADEMY MEMBERS WHO ATTENDED LECTURE COURSE

James E. Aiguier
Walter Brunk
C. H. Buck
Clarke E. Chamberlain
Ivan Rogers Cottrell
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Romle E. Romine
Carlos H. Schott
A. H. Schack
Grace Rogers Spalding
F. S. Tittle
Randall L. Westcott
Willa Yeretsky

Editor's Note: A Report of this Lecture Course will be published in the July issue of the *Journal of Periodontology*

"PATIENTS ARE DIET AND FLUORINE CONSCIOUS," SAYS ROBERT G. KESEL IN AN EDITORIAL IN A RECENT ISSUE OF FORTNIGHTLY REVIEW. * HE STATES FURTHER THAT:

"The search for a diet that will eliminate dental disease continues. There is abundant evidence that nutrition is associated with the two most prevalent dental disorders, caries and pyorrhea, but the manner of relationship is not yet understood.

"Caries has received the major attention because it affects so many individuals early in life (over ninety per cent of the civilized population). Many food elements, particularly various minerals and vitamins, have been stressed as all important, and numerous investigations have been reported which show a correlation between a deficiency of these elements and caries. Despite the extensive use of vitamin and mineral preparations caries and pyorrhea are as prevalent as ever. The fact that caries is so common permits the rather easy deduction that most everything is related to its etiology—and subjects to demonstrate the assumed association are readily found . . .

"Patients are becoming more and more diet conscious and are asking their dentists intelligent questions. Dentists must keep themselves informed if they are to avoid embarrassment and if they are to contribute their share to the clinical research that lies ahead."

* 7 (8): 13. April, 1944

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